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Lung Function, Airway Inflammation, and Polycyclic Aromatic Hydrocarbons Exposure in Mexican Schoolchildren:

A Pilot Study

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Abstract

Objective—To determine the association of exposure to polycyclic aromatic hydrocarbons (PAHs) with lung function and pH of exhaled breath condensate (EBC) in Mexican schoolchildren.

Methods—A pilot study was performed in a subsample of 64 schoolchildren from Mexico City. Lung function and pH of EBC were measured and metabolites of PAHs in urine samples were determined. The association was analyzed using robust regression models.

Results—A 10% increase in the concentrations of 2-hydroxyfluorene was significantly negatively associated with forced expiratory volume in 1 second (−11.2 mL, 95% CI: −22.2 to −0.02), forced vital capacity (−11.6 mL, 95% CI: −22.9 to −0.2), and pH of EBC (−0.035, 95% CI: −0.066 to −0.005).

Conclusion—Biomarkers of PAHs exposure were inversely associated with lung function and decrease of ph of EBC as a marker of airway inflammation in Mexican schoolchildren.

Polycyclic aromatic hydrocarbons (PAHs) constitute a group of compounds composed of fused benzenoid rings and formed by incomplete combustion or pyrolysis of organic materials. Significant PAHs sources include motor vehicle exhaust, residential and industrial

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heating, waste incineration, and tobacco smoke. The PAHs with low molecular weight (two- and three-ring) are mainly found in the gas phase and those with high molecular weight (five- and six-ring) are mainly associated with particles in air.[1] Smoking, grilling, and broiling lead to PAH formation in meat and in other foods. Considering the breadth and the variety of environmental sources, human exposure to PAHs occurs mostly through inhalation of polluted air and food consumption.[2] Measurement of urinary metabolites reflects recent exposure to PAHs, and each parent PAH can produce more than one measurable urinary metabolite. In addition, both naphthalene and the insecticide carbaryl are metabolized to 1-hydroxynaphthalene, making it difficult to distinguish between these exposures in the general population,[3] whereas only naphthalene metabolism results in 2-hydroxynaphthalene in urine. Pyrene is commonly found in PAH mixtures, and its urinary metabolite, 1-hydroxypyrene, has been used widely as an indicator of exposure to PAH chemicals, particularly by occupational exposure studies.[4]

The PAHs have received increased attention in recent years because some are carcinogenic or mutagenic. Although the lighter compounds have weaker carcinogenic/mutagenic properties, they are the most abundant in the urban atmosphere.[2] These compounds have also been associated with health outcomes unrelated to cancer, such as reproductive, cognitive, immunological, and respiratory effects.[5]

The Mexico City metropolitan area is a large polluted urban area with close to 85% of air pollutants coming from motor vehicles.[6] Although particulate matter, specifically the inhalable or respirable fraction (PM_{10} and $PM_{2.5}$), is routinely measured as an indicator of air pollution, PAHs are not frequently monitored and have been used much less as markers of air pollution.

Although exposure to fine particulates has been associated with fetal growth retardation, respiratory disorders, and cardiovascular disease, it is unknown whether PAHs contained in fine particulates are associated with these health effects.[5,7] In Mexico City, the health effects from particulate matter have been well documented,[8,9] but studies on exposure to PAHs and lung health have not been performed, to our knowledge. This study was undertaken to expand on our previous studies relating exposure to ambient air pollutants with respiratory health in children. We evaluated the association of urinary PAH metabolites with lung function and airway inflammation (measured as pH of exhaled breath condensate [EBC]) in these children. We hypothesized that acute exposure to PAHs is associated with decreased lung function and increased airway inflammation in this population.

Material and Methods

Study Design and Subjects

The children in the present cross-sectional pilot analysis were a subset (n = 64) of a larger cohort of 208 schoolchildren described previously.[10] The subset of children attended 37 of a total of 107 schools represented in the larger cohort.

Study Area

The study population consisted of schoolchildren living in three municipalities in the Mexico City metropolitan area with high levels of traffic-related emissions—Iztapalapa, Iztacalco, and Nezahualcoyotl.[6] The 64 schoolchildren included in the present analysis were recruited from clinics at the Federico Gomez Children's Hospital of Mexico (Hospital Infantil de México Federico Gómez), one of the largest pediatric hospitals in the city. The children were between 6 and 14 years old; they lived in the study area, attended public schools located close to their homes, were volunteers, and were not selected using probability-based sampling. All procedures were explained to the parents, who signed an informed consent form. The children also gave their informed assent. The study protocol was reviewed and approved by the ethics committees at both the National Institute of Public Health (INSP, Spanish acronym) and the Federico Gomez Children's Hospital of Mexico. The involvement of the Centers for Disease Control and Prevention (CDC) laboratory was determined not to constitute engagement in human subject research.

Data Collection and Health Outcomes

A general purpose questionnaire was used to collect information on sociodemographic variables, past health history, and potential second-hand tobacco smoke exposure at home. A respiratory symptoms questionnaire was given, and anthropometric measurements were taken at baseline. The questionnaire has been used in previous studies in Mexican population and include some questions derived from the ISAAC standardized questionnaire. Lung function was measured using an EasyOne spirometer (NDD Medical Technologies, Andover, Massachusetts) according to the American Thoracic Society standards and EBC was collected using an R-tube following American Thoracic Society/European Respiratory Society Task Force recommendations. Spirometric test and EBC sampling methods and validation processes have been described in detail in our previous reports.[8,11]

Exposure Assessment

First morning urine samples were collected from schoolchildren. Containers were provided to the parents and they were instructed on sample collection procedures. The urine samples were refrigerated at 4°C and were then aliquoted and frozen at −70°C until the time of analysis. The urine samples were analyzed for monohydroxy PAH metabolites[12] at the CDC. Creatinine concentrations were determined at the CDC laboratories by an enzymatic in vitro assay (Roche Diagnostic, Indianapolis, Indiana). Metabolites were reported as creatinine adjusted (ng/g creatinine). Urinary metabolite measurements in spot samples were routinely adjusted using creatinine to correct for urine concentration/dilution.[13]

Statistical Analyses

Geometric mean and percentiles for PAH biomarker levels were calculated. To compare subjects included in the analysis with those who were excluded, nonparametric tests on the equality of medians were performed. The associations of lung function and pH of EBC (dependent variables) with urinary PAH metabolite concentrations (independent variables) were analyzed by robust regression models. Models were run to evaluate exposure effects from the previous day. That is, the association was calculated by selecting the day that

pulmonary function was performed and assigning the urinary metabolite measurement from the previous day. Urinary PAH metabolites were log transformed to normalize the distribution. Models were adjusted for potential confounding factors including sex, age, second-hand smoke exposure at home, and body mass index. Other variables such as socioeconomic index (mother's education and school type), atopic status, the use of allergy medicine, diet, height, and season were not significant ($P > 0.10$) and did not alter the results by more than 1%. Also, different multipollutants models were tested; however, given the correlation between these measurements, we decided to present the results from a single pollutants model. Analyses were conducted using Stata software, version 10.1 (StataCorp LP, College Station, TX).

Results

Characteristics of participants versus nonparticipants are presented in Table 1. Fifty-nine percent were male, with a median age of 9.6 years, ranging from 6 to 13 years. The mean number of years for mothers' schooling was 8.4. Sixty percent of the children were exposed to second-hand tobacco smoke from an adult who smoked at home.

Urinary PAHs Biomarkers Data

Table 2 presents the descriptive statistics for the creatinine-adjusted concentrations of the nine monohydroxy PAHs—metabolites of fluorene, phenanthrene, pyrene, and naphthalene. Ninety-nine percent of the samples had levels higher than the limit of detection. Figures 1A to Figures 1B present a comparison of the PAH metabolite levels in this study with the results of 6- to 11-year-old children in the US National Health and Nutrition Examination Survey, which reported PAH biomarker levels from a statistically representative sampling of the US general population.[14] For most of the metabolites measured, the geometric mean was similar to that in the US National Health and Nutrition Examination Survey, except for 2-hydroxynaphthalene, 2-hydroxyfluorene, 9-hydroxyfluorene, and 2-hydroxyphenanthrene. The differences between these metabolites could not be explained by second-hand smoke exposure at home.

Association Between Health Outcomes and Urinary PAH Metabolites

The associations of urinary PAH biomarkers with lung functions and pH of EBC are shown in Table 3. We found a significant negative association of some urinary PAH metabolites with lung function and pH of EBC, after adjusting for age, sex, second-hand tobacco smoke, and body mass index. A 10% increase in the concentration of 2-hydroxyfluorene had a significant negative association with forced expiratory volume in 1 second (FEV_1) (−11.2 mL, 95% CI −22.2 to −0.02), forced vital capacity (FVC) (−11.6 mL, 95% CI −22.9 to −0.2), and pH of EBC (−0.035, 95% CI: −0.066 to −0.005) in schoolchildren. In the model for 1-hydroxyphenanthrene, we observed a marginally significant negative association with FEV_1 (−7.8 mL, 95% CI: −17.1 to 1.5, $P = 0.09$) and FVC (−9.1 mL, 95% CI: −19.9 to 1.7, $P = 0.09$). The other PAH metabolites measured showed a negative association trend with lung function, but these associations were not statistically significant.

Six out of the nine PAH metabolites were significantly associated with decreased pH of EBC, one was marginally associated, and 1-naphthal and 4-hydroxyphenanthrene were the only two metabolites that were not significantly associated with decreased pH of EBC (Table 3). For example, a 10% increase in 1-hydroxyphenanthrene concentration was associated with a decrease of −0.028 pH in EBC (−0.028, 95% CI: −0.047 to −0.010, P = 0.005).

Discussion

The results of this pilot study showed that acute exposure to PAHs, as evaluated by its urinary metabolites, had a negative association with pH of EBC and lung function in this cohort of Mexican schoolchildren. Although exposure to outdoor ambient levels of $PM_{2.5}$ and other air pollutants has been associated with decreased lung function, asthma, and respiratory symptoms in schoolchildren, this is the first study that explores an association with the inflammatory response and changes in lung function due to exposure to low-molecular-weight PAHs. A recently published study in Fresno, California, showed increased wheezing in asthmatic children exposed to PAHs, but this study measured air phenanthrene and 4-, 5-, and 6-ring particle-bound PAHs concentrations.[15] Phenanthrene was found to have an impact on wheezing and our results also show that phenanthrene metabolite (1-hydroxyphenanthrene) had a significant or marginally significant negative association with respiratory parameters.

Our results are also consistent with those reported in other studies relating PAH exposure to lung health. Certain hydrocarbons present in the environment, such as benzo(a)pyrene, can contribute to adduct formation in the lung. These adducts have occurred in bronchial epithelial cells and have been considered critical to effects on human lungs[16–18], however, we did not have information on this metabolite in our population.

Early animal studies on the influence of particles and PAHs exposure show that risks from the carcinogenicity of PAHs increase with prolonged retention of these compounds in the lungs because of lipophilic properties.[1] A major fraction (probably greater than 80%) of inhaled PAHs is expected to be deposited on the thin alveolar epithelium fraction and is likely to be retained in the lung tissue. Therefore, high local tissue concentrations can occur even with the low level of exposure to which humans are subjected, thereby inducing inflammatory processes.[1] These negative effects observed on lung function and the decrease of pH of EBC as marker of inflammatory response of airways are consistent with those reported in our previous studies on vehicular traffic-related air pollution, suggesting a short-term effect of particulate matter.

In our cohort study, $PM_{2.5}$ levels ranged from 3.1 to 96.1 $\mu g/m^3$ during the study period and were shown to affect both lung function and airway inflammation, evaluated by pH of EBC.[8,11] For the present analysis, we performed an objective evaluation of PAHs exposure, using multiple urinary biomarkers, and our results are consistent with those observed for exposure to local $PM_{2.5}$ concentration, for which we observed a reduction in FEV_1 of 21 mL for each increment in interquartile range of the 8-hour moving average of $PM_{2.5}$.[11]

Another contribution of this study is the evaluation of pH of EBC; Patel et al[19] recently reported that decreases in EBC pH, indicating increased airway inflammation. In this sense, we observed a significant negative association between pH of EBC in schoolchildren and increased levels of fluorene, phenanthrene, and naphthalene urinary metabolites. The results of this study for pH of EBC can be explained because exposure to PAHs has been shown to contribute to oxidative stress with increased secretions of proinflammatory cytokines and increases in the inflammatory response of the airways.[20] In vivo experimental studies of PAH exposure have reported apoptotic activity and its potential association with the inflammatory response, prolonged inflammation, and delay in repair processes of injured tissues.[21] Furthermore, Epton et al[20] evaluated the pH of EBC and exposure to PAHs in 93 male students but did not observe a significant effect of urinary 1-hydroxypyrene. In this study, 1-hydroxypyrene showed a significant association with decrease of pH of EBC, but not with lung function. Recently published results suggest that the pyrene metabolite has a large dietary component,[22] whereas other PAHs—such as fluorene, and to a lesser extent phenanthrene—may be more related to air pollution exposure. We found that fluorene and phenanthrene metabolites were more highly associated with respiratory disease parameters, which is consistent with the fact that air pollution is a main source of PAHs in our population. Meanwhile, 4-hydroxyphenanthrene and 1-hydroxynaphthalene were not associated with any respiratory parameters, probably because of very low concentrations of the former and potential pesticide exposure as a competing source of 1-hydroxynaphthalene.[3]

Some limitations must be taken into account when interpreting the results of this pilot study. First, we were only able to measure biomarkers for selected PAHs, while total PAH exposure includes large numbers of individual compounds in a given mixture. We could not evaluate exposure to high-molecular-weight PAHs, such as benzo[a]pyrene, because metabolites of the high-molecular-weight PAHs are difficult to detect in urine. Nevertheless, attention has recently focused upon the more abundant gas-phase PAHs, such as naphthalene, phenanthrene, and fluorene. Also, pyrene is normally abundant in PAH mixtures, and exposures occur through diet, polluted air, and cigarette smoke. In addition, 1-hydroxypyrene has been widely used as a representative biomarker of PAHs exposures.[14] Second, our sample size limited the power of this study, which directly affects the statistical significance of some associations and finally the possible bias by study design. Nonetheless, these preliminary results suggest a negative association of PAHs, as measured by urinary metabolites, with the inflammatory response and lung function.

Conclusion

Our preliminary results suggest that exposure to certain PAHs in the general environment may have an acute negative association on lung function and pH of EBC in a subset of Mexican schoolchildren. To our knowledge, this is the first study in showing an association between decreased lung function, decrease of Ph of EBC, and PAH exposure, evaluated with urinary biomarkers. Nevertheless, specifically designed epidemiological studies are needed to confirm the effect of PAH inhalation exposure on lung health and to clarify a plausible biological mechanism.

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All authors of this research paper have directly participated in the planning, execution, or analysis of this study. All authors of this paper have read and approved the final version submitted.

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The findings and conclusions in this report are those of the authors and do not necessarily represent the official position of the Centers for Disease Control and Prevention.

References

1. Bostrom CE, Gerde P, Hanberg A, et al. Cancer risk assessment, indicators, and guidelines for polycyclic aromatic hydrocarbons in the ambient air. Environ Health Perspect. 2002; 110(suppl. 3): 451–488. [PubMed: 12060843]
2. Srogi K. Monitoring of environmental exposure to polycyclic aromatic hydrocarbons: a review. Environ Chem Lett. 2007; 5:169–195.
3. Meeker JD, Barr DB, Serdar B, et al. Utility of urinary 1-naphthol and 2-naphthol levels to assess environmental carbaryl and naphthalene exposure in an epidemiology study. J Expo Sci Environ Epidemiol. 2007; 17:314–320. [PubMed: 16721410]
4. CDC Environmental Health. Fourth National Report on Human Exposure to Environmental Chemicals. Atlanta, GA: Centers for Disease Control and Prevention; 2009.
5. Toxicological profile for polycyclic aromatic hydrocarbons. Atlanta, GA: Agency for Toxic Substances and Disease Registry; 1995. Agency for Toxic Substances and Disease Registry.
6. Escamilla-Nuñez MC, Barraza-Villarreal A, Hernandez-Cadena L, et al. Traffic-related air pollution and respiratory symptoms among asthmatic children, resident in Mexico City: the EVA cohort study. Respir Res. 2008; 9:74. [PubMed: 19014608]
7. Choi H, Jedrychowski W, Spengler J, et al. International studies of prenatal exposure to polycyclic aromatic hydrocarbons and fetal growth. Environ Health Perspect. 2006; 114:1744–1750. [PubMed: 17107862]
8. Barraza-Villarreal A, Sunyer J, Hernandez-Cadena L, et al. Air pollution, airway inflammation, and lung function in a cohort study of Mexico City schoolchildren. Environ Health Perspect. 2008; 116:832–838. [PubMed: 18560490]
9. Rojas-Martinez R, Perez-Padilla R, Olaiz-Fernandez G, et al. Lung function growth in children with long-term exposure to air pollutants in Mexico City. Am J Respir Critical Care Med. 2007; 176:377–384. [PubMed: 17446338]
10. Romieu I, Barraza-Villarreal A, Escamilla-Nunez C, et al. Exhaled breath malondialdehyde as a marker of effect of exposure to air pollution in children with asthma. J Allergy Clin Immunol. 2008; 121:903.e6–909.e6. [PubMed: 18234317]
11. Barraza-Villarreal A, Escamilla-Nunez MC, Hernandez-Cadena L, et al. Elemental carbon exposure and lung function in school children from Mexico City. Eur Respir J. 2011; 38:548–552. [PubMed: 21310877]
12. Li Z, Romanoff LC, Trinidad DA, et al. Measurement of urinary monohydroxy polycyclic aromatic hydrocarbons using automated liquid-liquid extraction and gas chromatography/isotope dilution high-resolution mass spectrometry. Anal Chem. 2006; 78:5744–5751. [PubMed: 16906719]
13. Barr DB, Wilder LC, Caudill SP, et al. Urinary creatinine concentrations in the U.S. population: implications for urinary biologic monitoring measurements. Environ Health Perspect. 2005; 113:192–200. [PubMed: 15687057]
14. CDC Environmental Health. Third National Report on Human Exposure to Environmental Chemicals. Atlanta, GA: Center for Disease Control and Prevention; 2005.

15. Gale SL, Noth EM, Mann J, et al. Polycyclic aromatic hydrocarbon exposure and wheeze in a cohort of children with asthma in Fresno, CA. J Expos Sci Environ Epidemiol. 2012; 22:386–392.
16. Conney AH. Induction of microsomal enzymes by foreign chemicals and carcinogenesis by polycyclic aromatic hydrocarbons: G. H. A. Clowes Memorial Lecture. Cancer Res. 1982; 42:4875–4917. [PubMed: 6814745]
17. Feychting M, Svensson D, Ahlbom A. Exposure to motor vehicle exhaust and childhood cancer. Scand J Work Environ Health. 1998; 24:8–11. [PubMed: 9562395]
18. Liu M, Chen L, Zhou R, Wang J. Association between GSTM1 polymorphism and DNA adduct concentration in the occupational workers exposed to PAHs: a meta-analysis. Gene. 2013; 519:71–76. [PubMed: 23395936]
19. Patel MM, Chillrud SN, Deepti KC, et al. Traffic-related air pollutants and exhaled markers of airway inflammation and oxidative stress in New York City adolescents. Environ Res. 2013; 121:71–78. [PubMed: 23177171]
20. Epton MJ, Dawson RD, Brooks WM, et al. The effect of ambient air pollution on respiratory health of school children: a panel study. Environ Health. 2008; 14:16. [PubMed: 18479529]
21. Ferecatu I, Borot MC, Bossard C, et al. Polycyclic aromatic hydrocarbon components contribute to the mitochondria-antiapoptotic effect of fine particulate matter on human bronchial epithelial cells via the aryl hydrocarbon receptor. Part Fibre Toxicol. 2010; 7:18. [PubMed: 20663163]
22. Nethery E, Wheeler AJ, Fisher M, et al. Urinary polycyclic aromatic hydrocarbons as a biomarker of exposure to PAHs in air: a pilot study among pregnant women. J Expo Sci Environ Epidemiol. 2012; 22:70–81. [PubMed: 21915154]

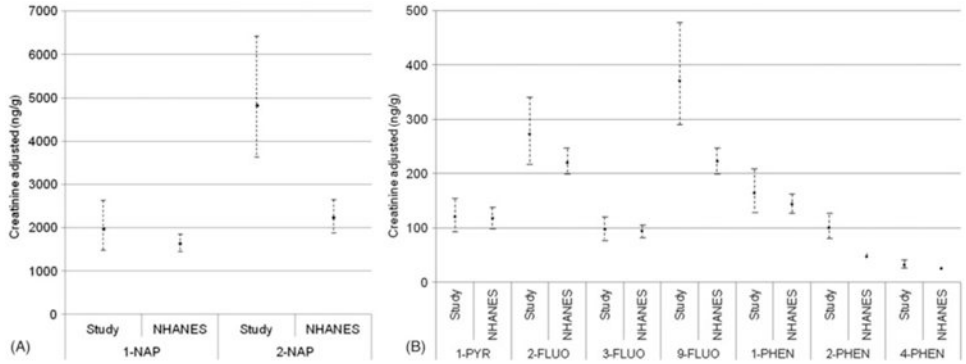

Figure 1.

(A) 1-Hydroxynaphthalene (1-NAP) and 2-hydroxynaphthalene (2-NAP) urinary metabolite concentration* for the Mexican study population† and children from the US National Health and Nutrition Examination Survey (NHANES) 2003 to 2004.‡ (B) PAHs urinary metabolite concentration* for the Mexican study population† and children from NHANES 2003 to 2004.‡ *Geometric mean (95% CI). †*N* = 64. ‡6 to 11 years. Data from CDC, Environmental Health, 2009.[4]

Table 1

Basic Characteristics and Main Outcomes of the Study Population (*N* = 64)

	Pilot Study (*N* = 64)		Large Cohort Study (*N* = 208)		
Variable	**Mean**	**SD**	**Mean**	**SD**	***P***
Sex (% male)	59.4		56.3		0.663
Age, yr	9.6	2.1	9.6	2.1	1.000
Weight, kg	36.4	11.4	36.8	12.6	0.821
Height, cm	136.7	13.0	136.2	13.9	0.799
Maternal schooling, yr	8.4	3.0	9.7	3.0	0.003
Second-hand tobacco smoke exposure at home, %	60.0		66.7		0.468
pH of exhaled breath condensate	7.71	0.47	7.46	0.50	0.001
FEV_1, L	1.89	0.58	1.91	0.65	0.826
FVC, L	2.35	0.68	2.29	0.77	0.576

FEV_1, forced expiratory volume in 1 second; FVC, forced vital capacity.

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Table 2

Monohydroxylated Polycyclic Aromatic Hydrocarbon Metabolite Urine Concentrations* in Schoolchildren† From Mexico City

PAHs Metabolites	Geometric Mean	95% CI	Percentile P_{50}	95% CI	Percentile P_{75}	95% CI
2-Hydroxyfluorene	274	219.6–340.7	279	229.5–363.4	424	75.1–549.4
3-Hydroxyfluorene	98	78.9–121.0	99	76.0–121.9	166	127.6–224.1
9-Hydroxyfluorene	372	290.1–477.0	366	275.9–492	673	506.7–954.4
1-Hydroxyphenanthrene	165	131.5–208.1	151	136.7–192.7	279	213.1–374.9
2-Hydroxyphenanthrene	101	80.9–125.3	107	81.8–127.9	158	129.4–204.1
4-Hydroxyphenanthrene	33	25.7–41.4	31	22.2–40.6	57	41.3–76
1-Hydroxypyrene	121	95.0–153.4	116	93.8–151	234	159.3–297.7
1-Hydroxynaphthalene	1977	1482.0–2637.6	2026	1327.4–2645	3712	2777.2–6266.7
2-Hydroxynaphthalene	4830	3635.0–6417.9	4721	3590.5–7202.9	9629	7521.4–15647.6

*ng/g creatinine.

†$N = 64$.

95% CI, 95% confidence interval.

Table 3

Association of PAH Metabolites* With pH of EBC and Lung Function in Schoolchildren From Mexico City

	pH of EBC		FEV_1, mL		FVC, mL	
PAHs Metabolites	**Coefficients† (95% CI)**	***P***	**Coefficients† (95% CI)**	***P***	**Coefficients† (95% CI)**	***P***
2-Hydroxyfluorene	−0.035 (−0.066, −0.005)	0.025	−11.22 (−22.20, −0.23)	0.046	−11.57 (−22.91, −0.24)	0.045
3-Hydroxyfluorene	−0.026 (−0.047, −0.005)	0.016	−7.18 (−15.81, 1.45)	0.101	−5.76 (−15.67, 4.15)	0.250
9-Hydroxyfluorene	−0.021 (−0.043, 0.000)	0.046	−6.48 (−13.52, 0.56)	0.071	−6.38 (−15.86, 3.10)	0.183
1-Hydroxyphenanthrene	−0.028 (−0.047, −0.010)	0.005	−7.80 (−17.06, 1.47)	0.097	−9.09 (−19.91, 1.74)	0.098
2-Hydroxyphenanthrene	−0.015 (−0.032, 0.002)	0.077	−2.76 (−10.70, 5.19)	0.490	−1.58 (−12.00, 8.85)	0.763
4-Hydroxyphenanthrene	−0.006 (−0.021, 0.009)	0.420	−3.02 (−12.15, 6.11)	0.510	−3.18 (−12.75, 6.39)	0.509
1-Hydroxynaphthalene	−0.015 (−0.036, 0.006)	0.159	0.25 (−8.39, 8.89)	0.954	−0.26 (−10.61, 10.09)	0.960
2-Hydroxynaphthalene	−0.011 (−0.021, 0.000)	0.042	−4.12 (−12.27, 4.03)	0.316	−4.27 (−13.11, 4.56)	0.337
1-Hydroxypyrene	−0.014 (−0.028, 0.000)	0.049	−6.26 (−14.04, 1.51)	0.112	−5.62 (−14.59, 3.34)	0.214

*LnPAHS, Log (PAHs metabolites).

†The coefficient is calculated for a 10% increase of each urine metabolite (ng/g creatinine). All models were adjusted for sex, age, second-hand tobacco smoke exposure at home, and body mass index.
CI, confidence interval; EBC, exhaled breath condensate; FEV_1, forced expiratory volume in 1 second; FVC, forced vital capacity; PAH, polycyclic aromatic hydrocarbon.